816,531 3/23/01

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=> s cytochrome(w) c

L9 53626 CYTOCHROME(W) C

=> s l9 and l2

L10 0 L9 AND L2

=> d l5 1-6 bib, ab

L5 ANSWER 1 OF 6 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2001:309278 BIOSIS
DN PREV200100309278
TI Genetic engineering of live rabies vaccines.
AU Morimoto, Kinjiro; McGettigan, James P.; Foley, Heather D.; Hooper, D. Craig; Dietzschold, Bernhard; Schnell, Matthias J. (1)
CS (1) Dorrance H. Hamilton Laboratories, Center for Human Virology, Thomas Jefferson University, 1020 Locust Street, Philadelphia, PA, 19107: matthias.schnell@mail.tju.edu USA
SO Vaccine, (14 May, 2001) Vol. 19, No. 25-26, pp. 3543-3551. print. ISSN: 0264-410X.
DT Article
LA English
SL English
AB **Rabies** virus is not a single entity but consists of a wide array of variants that are each associated with different host species. These viruses differ greatly in the antigenic makeup of their G proteins, the primary determinant of pathogenicity and major inducer of protective immunity. Due to this diversity, existing **rabies** vaccines have largely been targeted to individual animal species. In this report, a novel approach to the development of **rabies** vaccines using genetically modified, reverse-engineered live attenuated **rabies** viruses is described. This approach entails the engineering of vaccine **rabies** virus containing G proteins from virulent strains and modification of the G protein to further reduce pathogenicity. Strategies employed included exchange of the arginine at position 333 for glutamine and modification of the cytoplasmic domain. The recombinant viruses obtained were non-**neuroinvasive** when administered via a peripheral route. The ability to confer protective immunity depended largely upon conservation of the G protein antigenic structure between the vaccine and challenge virus, as well as on the route of immunization.

L5 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2000:441944 BIOSIS
DN PREV200000441944
TI Reinvestigation of the role of the rabies virus glycoprotein in viral pathogenesis using a reverse genetics approach.
AU Morimoto, Kinjiro; Foley, Heather D.; McGettigan, James P.; Schnell, Matthias J.; Dietzschold, Bernhard (1)
CS (1) Center for Neurovirology, Department of Microbiology and Immunology, Thomas Jefferson University, 1020 Locust Street, Philadelphia, PA, 19107 USA
SO Journal of Neurovirology, (October, 2000) Vol. 6, No. 5, pp. 373-381. print.
ISSN: 1355-0284.
DT Article
LA English
SL English
AB The **rabies** virus glycoprotein (G) gene of the highly **neuroinvasive** and neurotropic strains SHBRV-18, CVS-N2c, and CVS-B2c was introduced into the non-**neuroinvasive** and less neurotropic SN-10 strain to provide further insight into the role of G in the pathogenesis of **rabies**. Phenotypic analyses of the recombinant viruses revealed, as expected, that the neurotropism of a particular **rabies** virus strain was a function of its G. Nevertheless, the pathogenicity of the recombinant viruses was, in every case, markedly lower than that of the wild-type viruses suggesting that while the G dictates neurotropism, other viral attributes are also important in pathogenesis. The low pathogenicity of the recombinant viruses is at least in part due to a strong increase in transcription activity. On the other hand, the production of infectious virus by the R-SHB18 recombinant virus-infected cells was significantly delayed by comparison with SHBRV-18 wild-type virus infected-cells. Replacement of the R-SHB18 G cytoplasmic domain, transmembrane domain, and stem region with its SN-10 G counterparts neither results in a significant increase in budding efficiency nor an increase in pathogenicity. These results suggest that an optimal match of the cytoplasmic domain of G with the matrix protein may not be sufficient for maximal virus budding efficiency, which is evidently a major factor of virus pathogenicity. Our studies indicate that to maintain pathogenicity, the interactions between various structural elements of **rabies** virus must be highly conserved and the expression of viral proteins, in particular the G protein, must be strictly controlled.

L5 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1996:322441 BIOSIS
DN PREV199699044797
TI Characterization of a unique variant of bat rabies virus responsible for newly emerging human cases in North America.
AU Morimoto, Kinjiro; Patel, Menal; Corisdeo, Susanne; Hooper, D. Craig; Fu, Zhen Fang; Rupprecht, Charles E.; Koprowski, Hilary; Dietzschold, Bernhard (1)
CS (1) Center Neurovirol., Dep. Microbiol. Immunol., Thomas Jefferson Univ., 1020 Locust Street, Philadelphia, PA 19107-6799 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 11, pp. 5653-5658.
ISSN: 0027-8424.
DT Article
LA English
AB The silver-haired bat variant of **rabies** virus (SHBRV) has been identified as the etiological agent of a number of recent human **rabies** cases in the United States that are unusual in not having been associated with any known history of conventional exposure. Comparison of the different biological and biochemical properties of

isolates of this virus with those of a coyote street **rabies** virus (COSRV) reveal that there are unique features associated with SHBRV. In vitro studies showed that, while the susceptibility of neuroblastoma cells to infection by both viruses was similar, the infectivity of SHBRV was much higher than that of COSRV in fibroblasts (BHK-21) and epithelial cells (MA-104), particularly when these cells were kept, at 34 degree C. At this temperature, low pH-dependent fusion and cell-to-cell spread of virus is seen in BHK-21 cells infected with SHBRV but not with COSRV. It appears that SHBRV may possess an unique cellular tropism and the ability to replicate at lower temperature, allowing a more effective local replication in the dermis. This hypothesis is supported by in vivo results which showed that while SHBRV is less neurovirulent than COSRV when administered via the intramuscular or intranasal routes, both viruses are equally **neuroinvasive** if injected intracranially or intradermally. Consistent with the above findings, the amino acid sequences of the glycoproteins of SHBRV and COSRV were found to have substantial differences, particularly in the region that contains the putative toxic loop, which are reflected in marked differences in their antigenic composition. Nevertheless, an experimental **rabies** vaccine based on the Pittman Moore vaccine strain protected mice equally well from lethal doses of SHBRV and COSRV, suggesting that currently used vaccines should be effective in the postexposure prophylaxis of **rabies** due to SHBRV.

L5 ANSWER 4 OF 6 MEDLINE
AN 2001494966 MEDLINE
DN 21247205 PubMed ID: 11348722
TI Genetic engineering of live rabies vaccines.
AU Morimoto K; McGettigan J P; Foley H D; Hooper D C; Dietzschold B; Schnell M J
CS Department of Microbiology, Thomas Jefferson University, 1020 Locust Street, Philadelphia, PA 19107, USA.
NC AI 44340 (NIAID)
AI 45097 (NIAID)
SO VACCINE, (2001 May 14) 19 (25-26) 3543-51.
Journal code: X60; 8406899. ISSN: 0264-410X.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200109
ED Entered STN: 20010910
Last Updated on STN: 20010910
Entered Medline: 20010906
AB **Rabies** virus is not a single entity but consists of a wide array of variants that are each associated with different host species. These viruses differ greatly in the antigenic makeup of their G proteins, the primary determinant of pathogenicity and major inducer of protective immunity. Due to this diversity, existing **rabies** vaccines have largely been targeted to individual animal species. In this report, a novel approach to the development of **rabies** vaccines using genetically modified, reverse-engineered live attenuated **rabies** viruses is described. This approach entails the engineering of vaccine **rabies** virus containing G proteins from virulent strains and modification of the G protein to further reduce pathogenicity. Strategies employed included exchange of the arginine at position 333 for glutamine and modification of the cytoplasmic domain. The recombinant viruses obtained were non-**neuroinvasive** when administered via a peripheral route. The ability to confer protective immunity depended largely upon conservation of the G protein antigenic structure between the vaccine and challenge virus, as well as on the route of immunization.

L5 ANSWER 5 OF 6 MEDLINE
AN 2000479977 MEDLINE
DN 20486404 PubMed ID: 11031690
TI Reinvestigation of the role of the rabies virus glycoprotein in viral pathogenesis using a reverse genetics approach.
AU Morimoto K; Foley H D; McGettigan J P; Schnell M J; Dietzschold B
CS Department of Microbiology and Immunology, Thomas Jefferson University, Philadelphia, PA 19107, USA.
NC AI 41544 (NIAID)
AI 45097 (NIAID)
SO JOURNAL OF NEUROVIROLOGY, (2000 Oct) 6 (5) 373-81.
Journal code: CME. ISSN: 1355-0284.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200101
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010118
AB The **rabies** virus glycoprotein (G) gene of the highly **neuroinvasive** and neurotropic strains SHBRV-18, CVS-N2c, and CVS-B2c was introduced into the non-**neuroinvasive** and less neurotropic SN-10 strain to provide further insight into the role of G in the pathogenesis of **rabies**. Phenotypic analyses of the recombinant viruses revealed, as expected, that the neurotropism of a particular **rabies** virus strain was a function of its G. Nevertheless, the pathogenicity of the recombinant viruses was, in every case, markedly lower than that of the wild-type viruses suggesting that while the G dictates neurotropism, other viral attributes are also important in pathogenesis. The low pathogenicity of the recombinant viruses is at least in part due to a strong increase in transcription activity. On the other hand, the production of infectious virus by the R-SHB18 recombinant virus-infected cells was significantly delayed by comparison with SHBRV-18 wild-type virus infected-cells. Replacement of the R-SHB18 G cytoplasmic domain, transmembrane domain, and stem region with its SN-10 G counterparts neither results in a significant increase in budding efficiency nor an increase in pathogenicity. These results suggest that an optimal match of the cytoplasmic domain of G with the matrix protein may not be sufficient for maximal virus budding efficiency, which is evidently a major factor of virus pathogenicity. Our studies indicate that to maintain pathogenicity, the interactions between various structural elements of **rabies** virus must be highly conserved and the expression of viral proteins, in particular the G protein, must be strictly controlled.

L5 ANSWER 6 OF 6 MEDLINE
AN 96224342 MEDLINE
DN 96224342 PubMed ID: 8643632
TI Characterization of a unique variant of bat rabies virus responsible for newly emerging human cases in North America.
AU Morimoto K; Patel M; Corisdeo S; Hooper D C; Fu Z F; Rupprecht C E; Koprowski H; Dietzschold B
CS The Center for Neurovirology, Department of Microbiology and Immunology, Thomas Jefferson University, Philadelphia, PA 19107-6799, USA.
NC AI-09706 (NIAID)
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 May 28) 93 (11) 5653-8.
Journal code: PV3; 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199607
ED Entered STN: 19960726
Last Updated on STN: 19970203
Entered Medline: 19960717
AB The silver-haired bat variant of **rabies** virus (SHBRV) has been identified as the etiological agent of a number of recent human **rabies** cases in the United States that are unusual in not having been associated with any known history of conventional exposure. Comparison of the different biological and biochemical properties of isolates of this virus with those of a coyote street **rabies** virus (COSRV) revealed that there are unique features associated with SHBRV. In vitro studies showed that, while the susceptibility of neuroblastoma cells to infection by both viruses was similar, the infectivity of SHBRV was much higher than that of COSRV in fibroblasts (BHK-21) and epithelial cells (MA-104), particularly when these cells were kept at 34 degrees C. At this temperature, low pH-dependent fusion and cell-to-cell spread of virus is seen in BHK-21 cells infected with SHBRV but not with COSRV. It appears that SHBRV may possess an unique cellular tropism and the ability to replicate at lower temperature, allowing a more effective local replication in the dermis. This hypothesis is supported by in vivo results which showed that while SHBRV is less neurovirulent than COSRV when administered via the intramuscular or intranasal routes, both viruses are equally **neuroinvasive** if injected intracranially or intradermally. Consistent with the above findings, the amino acid sequences of the glycoproteins of SHBRV and COSRV were found to have substantial differences, particularly in the region that contains the putative toxic loop, which are reflected in marked differences in their antigenic composition. Nevertheless, an experimental **rabies** vaccine based on the Pittman Moore vaccine strain protected mice equally well from lethal doses of SHBRV and COSRV, suggesting that currently used vaccines should be effective in the postexposure prophylaxis of **rabies** due to SHBRV.

=> s neuroinvasive

L11 187 NEUROINVASIVE

=> s rabies

L12 14249 RABIES

=> s vaccine

L13 131470 VACCINE

=> s l11 and l12 and l13

L14 4 L11 AND L12 AND L13

=> d l14 1-4 bib, ab

L14 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2001:309278 BIOSIS
DN PREV200100309278
TI Genetic engineering of live **rabies** vaccines.
AU Morimoto, Kinjiro; McGettigan, James P.; Foley, Heather D.; Hooper, D. Craig; Dietzschold, Bernhard; Schnell, Matthias J. (1)
CS (1) Dorrance H. Hamilton Laboratories, Center for Human Virology, Thomas Jefferson University, 1020 Locust Street, Philadelphia, PA, 19107:

matthias.schnell@mai[redacted]tju.edu USA
SO Vaccine, (14 May, 2[redacted]) Vol. 19, No. 25-26, pp. 3543[redacted]51. print.
ISSN: 0264-410X.
DT Article
LA English
SL English
AB **Rabies** virus is not a single entity but consists of a wide array of variants that are each associated with different host species. These viruses differ greatly in the antigenic makeup of their G proteins, the primary determinant of pathogenicity and major inducer of protective immunity. Due to this diversity, existing **rabies** vaccines have largely been targeted to individual animal species. In this report, a novel approach to the development of **rabies** vaccines using genetically modified, reverse-engineered live attenuated **rabies** viruses is described. This approach entails the engineering of **vaccine rabies** virus containing G proteins from virulent strains and modification of the G protein to further reduce pathogenicity.
Strategies employed included exchange of the arginine at position 333 for glutamine and modification of the cytoplasmic domain. The recombinant viruses obtained were non-**neuroinvasive** when administered via a peripheral route. The ability to confer protective immunity depended largely upon conservation of the G protein antigenic structure between the **vaccine** and challenge virus, as well as on the route of immunization.

L14 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1996:322441 BIOSIS
DN PREV199699044797
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AU Morimoto, Kinjiro; Patel, Menal; Corisdeo, Susanne; Hooper, D. Craig; Fu, Zhen Fang; Rupprecht, Charles E.; Koprowski, Hilary; Dietzschold, Bernhard (1)
CS (1) Center Neurovirol., Dep. Microbiol. Immunol., Thomas Jefferson Univ., 1020 Locust Street, Philadelphia, PA 19107-6799 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 11, pp. 5653-5658.
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AN 2001494966 MEDLINE
DN 21247205 PubMed ID: 11348722
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AU Morimoto K; McGettigan J P; Foley H D; Hooper D C; Dietzschold B; Schnell M J
CS Department of Microbiology, Thomas Jefferson University, 1020 Locust Street, Philadelphia, PA 19107, USA.
NC AI 44340 (NIAID)
AI 45097 (NIAID)
SO VACCINE, (2001 May 14) 19 (25-26) 3543-51.
Journal code: X60; 8406899. ISSN: 0264-410X.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200109
ED Entered STN: 20010910
Last Updated on STN: 20010910
Entered Medline: 20010906
AB **Rabies** virus is not a single entity but consists of a wide array of variants that are each associated with different host species. These viruses differ greatly in the antigenic makeup of their G proteins, the primary determinant of pathogenicity and major inducer of protective immunity. Due to this diversity, existing **rabies** vaccines have largely been targeted to individual animal species. In this report, a novel approach to the development of **rabies** vaccines using genetically modified, reverse-engineered live attenuated **rabies** viruses is described. This approach entails the engineering of **vaccine rabies** virus containing G proteins from virulent strains and modification of the G protein to further reduce pathogenicity.
Strategies employed included exchange of the arginine at position 333 for glutamine and modification of the cytoplasmic domain. The recombinant viruses obtained were non-**neuroinvasive** when administered via a peripheral route. The ability to confer protective immunity depended largely upon conservation of the G protein antigenic structure between the
vaccine and challenge virus, as well as on the route of immunization.

L14 ANSWER 4 OF 4 MEDLINE
AN 96224342 MEDLINE
DN 96224342 PubMed ID: 8643632
TI Characterization of a unique variant of bat **rabies** virus responsible for newly emerging human cases in North America.
AU Morimoto K; Patel M; Corisdeo S; Hooper D C; Fu Z F; Rupprecht C E; Koprowski H; Dietzschold B
CS The Center for Neurovirology, Department of Microbiology and Immunology, Thomas Jefferson University, Philadelphia, PA 19107-6799, USA.
NC AI-09706 (NIAID)
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 May 28) 93 (11) 5653-8.
Journal code: PV3; 7505876. ISSN: 0027-8424.
CY United States

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199607
ED Entered STN: 19960726
Last Updated on STN: 19970203
Entered Medline: 19960717
AB The silver-haired bat variant of **rabies** virus (SHBRV) has been identified as the etiological agent of a number of recent human **rabies** cases in the United States that are unusual in not having been associated with any known history of conventional exposure. Comparison of the different biological and biochemical properties of isolates of this virus with those of a coyote street **rabies** virus (COSRV) revealed that there are unique features associated with SHBRV. In vitro studies showed that, while the susceptibility of neuroblastoma cells to infection by both viruses was similar, the infectivity of SHBRV was much higher than that of COSRV in fibroblasts (BHK-21) and epithelial cells (MA-104), particularly when these cells were kept at 34 degrees C. At this temperature, low pH-dependent fusion and cell-to-cell spread of virus is seen in BHK-21 cells infected with SHBRV but not with COSRV. It appears that SHBRV may possess an unique cellular tropism and the ability to replicate at lower temperature, allowing a more effective local replication in the dermis. This hypothesis is supported by in vivo results which showed that while SHBRV is less neurovirulent than COSRV when administered via the intramuscular or intranasal routes, both viruses are equally **neuroinvasive** if injected intracranially or intradermally. Consistent with the above findings, the amino acid sequences of the glycoproteins of SHBRV and COSRV were found to have substantial differences, particularly in the region that contains the putative toxic loop, which are reflected in marked differences in their antigenic composition. Nevertheless, an experimental **rabies** **vaccine** based on the Pittman Moore **vaccine** strain protected mice equally well from lethal doses of SHBRV and COSRV, suggesting that currently used vaccines should be effective in the postexposure prophylaxis of **rabies** due to SHBRV.

=> s dietzschold

L15 39 DIETZSCHOLD

=> s l12 and l15

L16 27 L12 AND L15

=> s l16 and l13

L17 24 L16 AND L13

=> l l17 and l14

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=> d l17 1-10 bib,

L17 ANSWER 1 OF 24 USPATFULL
AN 2001:152489 USPATFULL
TI Replication-defective adenovirus human type 5 recombinant as a

vaccine carrier
IN Ertl, Hildegund C[redacted]., Villanova, PA, United State[redacted]
Wilson, James M., Gladwyne, PA, United States
PA The Wistar Institute of Anatomy and Biology, Philadelphia, PA, United States (U.S. corporation)
The Trustees of the University of Pennsylvania, Philadelphia, PA, United
States (U.S. corporation)
PI US 6287571 B1 20010911
AI US 1999-347060 19990702 (9)
RLI Continuation of Ser. No. US 973233, now patented, Pat. No. US 6019978
Continuation of Ser. No. US 1995-461837, filed on 5 Jun 1995, now patented, Pat. No. US 5698202
PRAI US 1995-78 19950608 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Salimi, Ali R.
LREP Howson and Howson
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 24 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1077

L17 ANSWER 2 OF 24 USPATFULL
AN 2001:82315 USPATFULL
TI Recombinant multivalent viral **vaccine**
IN Scott, Fred W., Brooktondale, NY, United States
Ngichabe, Christopher K., Kikuyu, Kenya
Hu, Liangbiao, Baltimore, MD, United States
Esposito, Joseph J., Atlanta, GA, United States
PA Cornell Research Foundation, Inc., Ithaca, NY, United States (U.S. corporation)
The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 6241989 B1 20010605
AI US 1995-552369 19951103 (8)
RLI Continuation-in-part of Ser. No. US 1994-190789, filed on 27 Jan 1994, now abandoned Continuation of Ser. No. US 1991-726609, filed on 9 Jul 1991, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Scheiner, Laurie
LREP Hodgson, Russ, Andrews, Woods & Goodyear LLP
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1211
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 3 OF 24 USPATFULL
AN 2001:14613 USPATFULL
TI Synthetic peptides for rubella **vaccine**
IN Chong, Pele, Richmond Hill, Canada
Gillam, Shirley, Vancouver, Canada
Ou, Dawei, Vancouver, Canada
Tingle, Aubrey, Vancouver, Canada
PA Connaught Laboratories Limited, Toronto, Canada (non-U.S. corporation)
PI US 6180758 B1 20010130
AI US 1997-834130 19970414 (8)
RLI Continuation of Ser. No. US 1994-256747, filed on 6 Oct 1994, now patented, Pat. No. US 6037448
DT Utility
FS Granted
EXNAM Primary Examiner: Stucker, Jeffrey
LREP Sim & McBurney

CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1559
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 4 OF 24 USPATFULL
AN 2000:134588 USPATFULL
TI Viral ribonucleocapsid as an immunological enhancer
IN Hooper, Douglas Craig, Medford, NJ, United States
Dietzschold, Bernhard, Newtown Square, PA, United States
Koprowski, Hilary, Wynnewood, PA, United States
PA Thomas Jefferson University, Philadelphia, PA, United States (U.S. corporation)
PI US 6129921 20001010
AI US 1995-567713 19951205 (8)
RLI Continuation of Ser. No. US 1994-230158, filed on 19 Apr 1994, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Allen, Marianne P.; Assistant Examiner: Zeman, Mary K
LREP Seidel, Gonda, Lavorgna & Monaco, PC
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 430
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 5 OF 24 USPATFULL
AN 2000:37390 USPATFULL
TI Polypeptides fused with alfalfa mosaic virus or ilarvirus capsid proteins
IN Koprowski, Hilary, Wynnewood, PA, United States
Yusibov, Vidadi, Havertown, PA, United States
Hooper, Douglas Craig, Medford, NJ, United States
Modelska, Anna, Wynnewood, PA, United States
PA Thomas Jefferson University, Philadelphia, PA, United States (U.S. corporation)
PI US 6042832 20000328
AI US 1996-704856 19960828 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Stucker, Jeffrey; Assistant Examiner: Bui, Phuong T.
LREP Volpe and Koenig, PC
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 1017
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 6 OF 24 USPATFULL
AN 2000:31521 USPATFULL
TI Synthetic peptides for a rubella **vaccine**
IN Chong, Pele, Richmond Hill, Canada
Gillam, Shirley, Vancouver, Canada
Ou, Dawei, Vancouver, Canada
Tingle, Aubrey, Vancouver, Canada
PA Connaught Laboratories Limited, North York, Canada (non-U.S. corporation)
PI US 6037448 20000314
WO 9314206 19930722
AI US 1994-256747 19941006 (8)
WO 1993-CA14 19930120
19941006 PCT 371 date
19941006 PCT 102(e) date

PRAI GB 1992-1139 19920120
DT Utility
FS Granted
EXNAM Primary Examiner: Nucker, Christine M.; Assistant Examiner: Stucker, Jeffrey
LREP Sim & McBurney
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 2538
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 7 OF 24 USPATFULL
AN 2000:18041 USPATFULL
TI **Vaccine** against **rabies** and process for preparation thereof
IN Lathe, Richard, Strasbourg, France
Kieny, Marie-Paule, Strasbourg, France
Drillien, Robert, Strasbourg, France
Lecocq, Jean-Pierre, Reichsteet, France
PA Transgene S.A., Strasbourg, France (non-U.S. corporation)
PI US 6024953 20000215
AI US 1994-231457 19940421 (8)
RLI Continuation of Ser. No. US 1993-38052, filed on 29 Mar 1993, now abandoned which is a continuation of Ser. No. US 1991-759138, filed on 11 Sep 1991, now abandoned which is a continuation of Ser. No. US 1989-378801, filed on 11 Jul 1989, now abandoned which is a continuation of Ser. No. US 1985-829144, filed on 24 Dec 1985, now abandoned
PRAI FR 1984-6499 19840425
WO 1985-FR96 19850424
DT Utility
FS Granted
EXNAM Primary Examiner: Degen, Nancy; Assistant Examiner: Schwartzman, Robert
LREP Burns, Doane, Swecker & Mathis, L.L.P.
CLMN Number of Claims: 9
ECL Exemplary Claim: 7
DRWN 12 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 709

L17 ANSWER 8 OF 24 USPATFULL
AN 2000:12441 USPATFULL
TI Replication-defective adenovirus human type 5 recombinant as a **vaccine** carrier
IN Ertl, Hildegund C. J., Villanova, PA, United States
Wilson, James M., Gladwyne, PA, United States
PA The Wistar Institute of Anatomy and Biology, Philadelphia, PA, United States (U.S. corporation)
The Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
PI US 6019978 20000201
WO 9639178 19961212
AI US 1997-973223 19971203 (8)
WO 1996-US9495 19960605
19971203 PCT 371 date
19971203 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1995-461837, filed on 5 Jun 1995, now patented, Pat. No. US 5698202
PRAI US 1995-78 19950608 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Mosher, Mary E.; Assistant Examiner: Salimi, Ali R.
LREP Howson and Howson
CLMN Number of Claims: 6

ECL Exemplary Claim: 1
DRWN 4 Drawing Figure([redacted] 6 Drawing Page(s)
LN.CNT 1435
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 9 OF 24 USPATFULL
AN 1998:143659 USPATFULL
TI Method for generating an immunogenic T cell response protective against a virus
IN Heber-Katz, Ellen, Philadelphia, PA, United States
Dietzschold, Bernhard, Newtown Square, PA, United States
PA The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)
PI US 5837249 19981117
AI US 1993-139609 19931020 (8)
RLI Continuation of Ser. No. US 1992-868946, filed on 15 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-685459, filed on 12 Apr 1991, now abandoned which is a continuation of Ser. No. US 1987-47443, filed on 8 May 1987, now abandoned which is a continuation-in-part of Ser. No. US 1985-725087, filed on 19 Apr 1985, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Woodward, Michael P.
LREP Banner & Witcoff, Ltd.
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1114
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 10 OF 24 USPATFULL
AN 1998:134638 USPATFULL
TI **Vaccine** against **rabies** and process for preparation thereof
IN Lathe, Richard, Strasbourg, France
Kieny, Marie-Paule, Strasbourg, France
Drillien, Robert, Strasbourg, France
Lecocq, Jean-Pierre, Reichsteet, France
PA Transgene S.A., Strasbourg, France (non-U.S. corporation)
PI US 5830477 19981103
AI US 1995-480736 19950607 (8)
RLI Continuation of Ser. No. US 1994-231457, filed on 21 Apr 1994 which is a
continuation of Ser. No. US 1993-38052, filed on 29 Mar 1993, now abandoned which is a continuation of Ser. No. US 1991-759138, filed on 11 Sep 1991, now abandoned which is a continuation of Ser. No. US 1989-378801, filed on 11 Jul 1989, now abandoned which is a continuation-in-part of Ser. No. US 1985-829144, filed on 24 Dec 1985, now abandoned
PRAI FR 1984-6499 19840425
DT Utility
FS Granted
EXNAM Primary Examiner: Degen, Nancy
LREP Burns, Doane, Swecker & Mathis, L.L.P.
CLMN Number of Claims: 36
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 988

=> s hiv or (human immunodeficiency virus)

L18 245429 HIV OR (HUMAN IMMUNODEFICIENCY VIRUS)